the remaining abscess) and seven were so-called interim operations.

All patients not immediately operated on were treated by the Ochsner method; that is, they were kept in bed and given no food or medicine by mouth as long as any decided tenderness was present. In eight cases rectal feeding was carried out for several days. Stomach lavage was employed in only one case. Ice was applied to the abdomen in one case and hot-water bag in several. Opiates, usually morphia, were given cautiously but sufficient to secure reasonable relief from pain. Cathartics were not given until gas was passing freely and the bowels had been moved by enema. In one case of general peritonitis, distention was so great that an aspirating needle was passed through the abdominal wall three or four different times and gas and liquid feces drawn off. The appendix was removed in all these except the abscess cases. All appendices removed presented evidence of constrictions. In only two were true concretions found. Both of these were distended and strangulated. One was wholly gangrenous in an early stage, and the other presented two necrotic spots, one of which broke down within an hour of its removal. The only complications encountered were a suture abscess late in the second week in a patient suffering from pulmonary tuberculosis, and a fecal fistula in one of the late operations for abscess. Besides these 28 cases, I have during these three years seen 16 patients in consultation. Most of these were treated conservatively, though many of them were given food and medicine by mouth during the acute "peritonitis" stage. Only one was operated upon during the early acute stage, three during the interim and three late in the attack, opening the remaining abscess.

There were two deaths. The first of these was a young woman about 34, suffering from pulmonary tuberculosis in an advanced stage. I saw her only once in consultation, about six days after the beginning of the attack. She then had a general peritonitis, of which she died five or six days later. She was not treated wholly by the Ochsner plan, as she was given considerable food and medicine by the mouth. The second case was seen in consultation just about 48 hours after the onset of the attack. There was evidence that perforation had just taken place, but I concurred in the opinion that probably it was so recent that immediate operation would still remove all infection. However, the patient was moved about seven miles to the County Hospital, and then subjected to operation. There was considerable free pus in the abdomen. The patient did badly under the ether, and died in 24 hours from general infection. This patient should not have been operated upon, and in a similar case I should object in future. This case is one of the first in this series, and I had not yet become so fully convinced that some of the infection is bound to remain and cause mischief in these cases. In all of these cases the idea of obstruction and distention as the cause of appendicitis has been acted upon, and has been a valuable aid. The Ochsner plan of treatment has been generally advised, and in my own cases fairly carried out; in many of the consultation cases, only partially at best.

The cases are presented as a plea for the Ochsner plan of treatment. All the patients so treated recovered. The entire series, 44 cases with two deaths, a mortality of a little over 4%, certainly emphasizes the advantage of avoiding operation during the acute stage, and altogether makes a strong argument for the conservative treatment of appendicitis, while acute peritonitis is present.

Conclusions.—1. Obstruction and consequent distention and strangulation is the cause of appendicitis.

2. The exact diagnosis can usually be made from a careful study of the history.

3. The ideal treatment is removal of the appendix before the infection extends to the peritoneum.

4. After this time no operation should be done during the acute stage.

5. Fasting, avoidance of cathartics and absolute rest insures a very low percentage of mortality in appendicitis and peritonitis.

[For discussion see the JOURNAL for October, 1904, pages 300 *et seq.*]

MYOCARDITIS, WITH SPECIAL REFERENCE TO DISORDERED METABOLISM.

By DR. W. W. KERR, San Francisco.

(*Continued from page* 371, *Vol.* II.)

The next question is, What is the cause of the myocardial change? Naturally the first inclination is to ascribe it to anemia from hemorrhage, but it frequently occurs where there was neither hemorrhage nor profuse menstruation, as in the case just quoted; and so this explanation will not suffice. Certainly it is possible that anemia may result in such cases independent of hemorrhage, but this itself would simply be an evidence of disturbed metabolism which would be liable to produce changes in other tissues as well as in the blood. It has not been in my power to obtain a sufficient number of blood counts to warrant any conclusion as to the general appearance of the blood in this class of cases, but the following case will show that the anemia is not necessarily very great. In this instance the left ventricle was dilated, and a systolic murmur could be heard in the mitral and tricuspid areas as well as over the base of the heart in the second left interspace. An examination of the blood made two hours before the tumor was removed gave the following count: Red cells, 4,700,000; leukocytes, 8,000; blood platelets very numerous; hemoglobin, 66%; color index, 0.70. General appearance of cells normal. No parasites found. Differential count, eosinophiles 0.0%, large mononuclears 1.5%, lymphocytes 17.5%, neutrophiles 81%. This patient had not suffered from hemorrhage, nor did the tumor give her any inconvenience, although of considerable size; but she had it removed simply because she felt worried by the knowledge that a growth was present. The only sign of heart disturbance of which she complained was that she became dyspneic more readily upon exertion.

Examination of the heart four weeks after the operation showed that the murmurs had disappeared from the base and tricuspid area, that the mitral sound was impure, and all the cardiac sounds of less intensity than the average.

Kessler in commenting upon his case, of which an abstract has been presented, offers a theory that places the cardiac changes upon a somewhat physical basis. He believes that it is not so much the size of the tumor as its consistency that is responsible for the trouble, and just as the heart, especially the left ventricle hypertrophies during pregnancy, so do similar changes take place in patients suffering from fibroid tumors of the uterus; but they will be better marked in the latter condition, because during pregnancy the increased work of the heart is simply the result of physiological increase in the vascular area, comparatively temporary in character, as it ceases with partruition; but since the tumor consists of dense nodules with a tense capsule, it presents a marked resistence to the circulation, which is also persistent, and consequently the strain upon the heart is both greater and more prolonged. Experience makes one naturally suspicious of a theory that puts a morbid process upon such a mechanical basis. Although it would account for the improvement in the heart, which sometimes follows removal of the tumor, and even if a parallel has been drawn between this and the transitory hypertrophy of pregnancy we must

remember that all are not agreed in conceding the exertion of propelling the blood through an increased capillary to be the cause of the enlargement, but that many regard hypertrophy as secondary to an increased effort necessary for the elimination of an increased amount of waste matter associated with the growth of the fetus.

Another suggestion is that the fibroids and the myocardial changes are due to a common cause. The tumors form in relation to the walls of the blood vessels in the uterus, and it is possible that a simultaneous change takes place in the muscular branches of the coronary arteries, and results in the formation of connective tissue in the cardiac muscle. Upon such a theory as this there cannot be any reason for improvement in the condition of the heart after operation, and yet we know positively that improvement does take place in some cases.

Fleck, in eighty examinations, found that a diseased ovary coexisted with the fibroid, and suggested that the myoma and myocardial changes were both due to an abnormal internal secretion from a diseased ovary. It would be interesting to know what particular disease of the ovary yields this abnormal secretion, since there are so many ovarian troubles without fibroids, and in operations, when the heart has improved after the removal of a fibroid, it should be stated whether the ovary was taken away at the same time.

This whole matter is one which requires very much closer study and compilation of statistics before any satisfactory conclusion can be obtained. It is only within the last few years that any special relationship has been claimed to exist between the uterine tumor and the cardiac changes, and consequently most of the histories lack precision in their description of the cardiac symptoms, the time of their appearance compared with that of the tumor, the condition of the blood in each case, the presence or absence of hemorrhage, the amount of menstrual flow, and the variety of fibroid with which the cardiac symptoms are most frequently associated, *i. e.*, whether it is subperitoneal, mural, or submucus; the fact that there are very many women who have fibroid growths in the uterus, and who do not appear to suffer any inconvenience, is rather puzzling; but it may be that if each one were questioned about the existence of palpitation or a growing tendency to dyspnea upon ordinary exertion, those symptoms would be discovered of which the patient did not complain because she did not suspect any relation between them and the uterine condition; furthermore, if the physical theory of increased resistence to the circulation be true, it is very likely that the situation of the tumor will have some effect in determining the symptoms, because the obstruction to the flow of blood through the uterine vessels will be greater at some points than at others.

The size of the growth is not entirely responsible for the cardiac changes, because in the first case that I quoted the cardiac asthenia was well marked before the fibroid had attained the size of a hen's egg, and this seems to have been the experience of others, who state that the cardiac symptoms frequently antedate the presence of a tumor that can be detected by any method of examination. But although the etiology of this condition is practically unknown, there are more lessons of importance to be obtained from the knowledge that such a relationship exists. First, in all cases of obscure persistent cardiac asthenia the possibility of a uterine origin should suggest itself. Second, when cardiac weakness and uterine fibroids coexist, unsuccessful medication should not be persisted in after a fair trial has been given, because tissue changes in the heart may take place to an extent beyond recovery. A woman should not be told that because her fibroid is not so large as to give her discomfort from pressure, or of such a nature as to debilitate her by hemorrhage, that she is perfectly safe to leave it alone, and that it will disappear with the menopause.

Statistics are imperfect as to the amount of improvement that takes place after the removal of the fibroid, but there cannot be any doubt that in many instances great benefit results; in fact, my attention was first attracted to the subject by the statement of the patient that since the operation her heart has ceased to trouble her. On the other hand operative treatment may have been delayed so long that the myocardium is changed to such an extent as to render any marked improvement impossible, and to increase the danger of the operation so as to render it unjustifiable.

The last series of cases includes those specific infectious diseases which are frequently associated with or followed by myocardial changes. In all febrile infectious diseases the myocardium is liable to suffer simply from the effects of prolonged high temperature; but on the other hand toxic products of certain microorganisms seem to have a special affinity for the cardiac muscle, or it may be that these specific agents, although themselves not very harmful, facilitate the growth of secondary infections which are highly injurious to the heart; or it may be that the microorganisms themselves are present in the myocardium. In cases of pneumonia, rheumatism, scarlet fever, enteric and several others, the physician is continually on the outlook for cardiac complications during the active stage of the disease, but it frequently happens that symptoms of myocardial changes do not appear until the patient is well advanced in convalescence, or even has been discharged as cured, and this late appearance of the symptoms may be attributed to one or more of several conditions. Thus the myocardium may have been injured during the acute stage, but only exhibited symptoms under the strain of exercise or exertion, or it may be that we have the influence of the toxins continuing after the acute stage has passed, just as we have post-diphtheritic paralysis appearing many weeks after all trace of the local infection has disappeared; or it is possible, indeed certain, that during these infectious processes the arterial system, including the arteries, becomes involved, and subsequently we have the gradual degeneration of the myocardium secondary to the coronary changes which took place during the acute infection.

The present intention is to refer only to three of the many infectious diseases associated with myocardial disturbance, and these have been selected because they probably are the most common on this coast, and also for the reason that they illustrate better than any others the appearance of cardiac symptoms during convalescence, or when the patient was supposed to have gained perfect health. The diseases are smallpox, influenza and typhoid fever.

Smallpox.—The following case is fairly illustrative of what may occasionally be found in patients who have suffered from smallpox. On the morning of July 6th, 1883, I was called to see Mrs. A., age 29, who was said to have been "in a fit" for nearly two hours. The patient was lying in bed, unconscious, her eyes open and staring, with the pupils widely and equally dilated. Her body was cold. The radial pulse was slow, irregular and poorly filled; her first sound of the heart could not be heard, the second was loose and flapping in character. At times the breathing appeared to have ceased, and then there would be two or three long respiratory movements. The patient was drawn to the edge of the bed so that her head and shoulders hung over it, with the result that the pupils at once began to diminish in size, breathing became more natural, and in less than two minutes consciousness returned. By this time nitrate of amyl had been obtained, and shortly after inhaling one pearl the patient fell into a sound natural sleep. At my next visit the following history was obtained: Until within the last three years the patient never suffered from any sickness, and had followed her occupation of trapeze performer; but at that time she had a very severe attack of smallpox, after which she suffered from symptoms of heart disease that prevented her resuming her professional career. These symptoms consisted of a feeling of weight and oppression over the precordia sometimes amounting to actual pain, palpitation and shortness of breath upon exertion, and occasional attacks of vertigo. Her extremities were al-

ways cold; she noticed that her ankles were somewhat swollen when she was going to bed at night, but in other respects she enjoyed perfect health. Examination failed to detect the apex beat, either upon inspection or palpitation, the area of cardiac dullness was not increased, the intensity of all the heart sounds was diminished, the first being inaudible in the mitral area.

The diagnosis made in this case was fatty heart secondary to smallpox. In smallpox endocarditis and pericarditis are rare, but in severe forms, especially the confluent variety, it is not uncommon to meet with myocardial changes, both fatty and parenchymatous. These may be due to the toxins of the disease or to secondary infections during maturation, and as a rule recovery takes place after a comparatively short time; but in this case the condition was more persistent, and continued to increase in severity during the first year after the sickness; so it is possible that the degeneration was secondary to endoarteritis of the coronary vessels, a change which French pathologists have described in connection with smallpox.

This patient took arsenic for nearly four months, and improved so much that she was free from pain and able to do ordinary domestic work; but, being a widow with matrimonial aspirations, she very shortly afterward married into her own rising profession, and thus I was robbed of the opportunity of furnishing you with the further history of the case.

Influenza.—Everyone is familiar with the intense general depression consequent upon influenza, and also with the great number of sequelæ consequent upon this disease; indeed so many conditions of poor health appear to date from an attack of grippe that occasionally one is almost ashamed to state his opinion in case it appears to be a cloak of ignorance; nevertheless, it is very questionable whether there is any other malady with such brief primary symptoms that is associated with so much general depression and so frequently paves the way for serious secondary infections. More than sixty years ago Sir Henry Holland called attention to the long-persisting effects of this disease upon those who had suffered from it, so we may comfort ourselves with the reflection that even in the good old days when opinions were formed to a much greater extent from personal observations and the minds of men were less liable to be biased by a plethora of medical journalism it was recognized that influenza was responsible for a multitude of evils.

The following case came under my observation last winter, and is quoted because it is a fair sample of the insidious way in which myocarditis frequently follows an attack of influenza:

Mr. G., age 43, came to my office during the first week in January, with the statement that he did not feel that there was anything especially the matter, that here was not any organ that seemed to have gone wrong, no palpitation, no indigestion, but that he was out of sorts, and that several friends had commented upon his gray appearance. Upon examination the pulse was between 120 and 130, the apex was three-quarters of an inch outside of the left nipple line, and a loud blowing systolic murmur was heard in the mitral area. In answer to an inquiry as to whether there had been any trouble with his heart, he stated that this was the first time he had ever consulted a physician, so he could not say, but that his custom from boyhood had been to spend two months in the mountains every year hunting game, and that he never had suffered any inconvenience. He further stated that just before Christmas he had an attack of influenza, so mild that, although he felt miserable, he did not remain at home during the day, and about one week later, when running up a hill to overtake a car, he experienced a peculiar sensation about his heart that made him give up the attempt. With rest in bed for a month, and the use of iron and strychnin, the heart recovered its normal size, and the murmurs disappeared, but it is still very irritable, so that any excitement induces a prolonged attack of palpitation.

This is the kind of history that is most frequently found in cases of myocarditis whose origin can be traced to grippe. It seems that cardiac disturbance more frequently follows the mild than the acute attacks, possibly for the reason that the patient is less careful of himself, and no precautions are taken against the sequels that are liable to ensue. Another point of importance is that myocarditis is much more common than endocarditis after influenza; at least such has been my experience. Such being the case, we can readily understand that a weak muscle may be readily overlooked, and the patient allowed to resume work when an endocardial murmur would have been detected immediately, and as a result the patient detained in bed for a considerable length of time; the presence or absence of a bruit seems to appeal to some men more forcibly than the quality of pulse, and consequently the condition of the cardiac muscle is not always so thoroughly investigated as it should be in patients suffering from influenza; so it may be that the true state of affairs only becomes manifest after the patient has resumed his daily duties and the ventricular wall yields under the improved strain. It is sometimes necessary, however, in this disease to make some allowance for the effects of medicine when estimating the condition of the myocardium, because influenza is very frequently treated by the administration of some of the coal tar series without the addition of some cardiac stimulant to guard against the depression which most of these substances produce. The influenza epidemic of 1889 was probably the most severe that this city has experienced, and it has often been a question with me whether the mortality was not materially increased by the use of antipyrin, which was the most popular remedy both in this country and in Europe at that time. The average dose was ten grains every four hours, and people purchased and used the powders without the advice or supervision of a physician.

On the other hand it must be admitted that the toxins of influenza remain active long after the acute symptoms have disappeared, and are capable of producing new symptoms; the intense inertia of mind and body that persists for weeks, it may be months, bear witness to the truth of this statement, while the subsequent appearance of both sensory and motor neuritis testify to long-continued activity of the poison. One cannot read the history of influenza epidemics without being impressed with the similarity between it and diphtheria; both diseases begin suddenly, and show a preference for the respiratory tract; the sequelæ are not in proportion to the severity of the primary attack, and the late appearance of those aftereffects suggests that in both diseases the toxins elaborated during the life of the microorganism and absorbed into the circulation are slow in elimination, and retain their violence for a long time.

Typhoid Fever.—Just as certain drugs have a special affinity for the heart, so have certain microorganisms, and among those may be included the bacillus of typhoid fever. Even as early as the end of the stage of incubation, signs of cardiac weakness may be detected, and during the first week of the fever, before pyrexia has existed long enough to do any harm, the dicrotic pulse is often one of the earliest symptoms that awakens suspicion regarding the exact nature of the disease. So marked is this early depression in some cases that French writers describe a cardiac variety of typhoid in which the fever is low or absent but the pulse is small, weak and irregular from the beginning; and yet even more positive evidence of the relationship between enteric involvement and changes in the heart muscle has been furnished by the discovery of the bacilli in the myocardium. The injury inflicted by this microorganism is not limited to the muscular fibres, but extends to the interstitial tissue and blood vessels, so that we not only have myocardial degeneration, but there may also be intermuscular infiltration with cells, and the small arteries blocked by endoarteritis.

The lesions produced in the myocardium may persist during and after convalescence, giving rise to attacks of extreme cardiac weakness much more pronounced than was expected from the condition of affairs during the acute stage; on the other hand, there is less reason to believe that the disease of the aorta and arteries initiated during an attack of typhoid

fever may become progressive and only manifest symptoms some years later.

In 1891 I saw in consultation a male nurse who had passed through a comparatively mild attack of enteric fever. The afternoon during his convalescence, while walking in the garden, the patient suddenly fell to the ground in an unconscious condition, and it was only after repeated injections of strychnia and the maintenance of artificial respiration continuously for more than three hours that he roused. During the attack the heart action and respirations were very feeble, but prior to this time his pulse had been good, and trouble was not anticipated. Forty-eight hours later the pulse and respirations were apparently normal. There was a second attack two weeks after the first, which was not so severe, but after this the patient progressed favorably, and at the end of three months went home to his friends in the eastern states, apparently in good health.

There was considerable discussion among the different physicians who saw this patient as to the pathology of the case, most of them being inclined to the opinion that it was one of fatty heart consequent upon the fever; but the apoplectiform character of the seizure, its long duration, the good quality of the pulse, both before and soon after the attack, have always made me suspect that we were dealing with an embolism of one of the smaller branches of the coronary artery derived from a thrombotic deposit upon the roughened lining of the vessel. Osler's emphatic statement that "sudden death not infrequently follows the block of one of the branches of the coronary artery, etc., etc., and this condition may constitute the sole lesion except a slight arterio-sclerosis," is a warning that should always be borne in mind.

But it may be that the symptoms of cardiac disturbance do not become apparent until some years after the patient has suffered from enteric fever. In 1896 I saw a man who was suffering from his first attack of angina pectoris, and there was not anything in his habits or history that could account for the disease except a severe attack of typhoid fever in the year 1890. I know personally that until that time his heart was perfectly sound, but afterward he never regained his former vigor. Subsequently he developed a slight dilatation of the heart with muscular mitral incompetence, and died three years later. No autopsy was obtained. This case is mentioned, not with the intention of demonstrating the relationship between typhoid fever and late cardiac disturbance, but rather with the view of illustrating the observations made by Landowzy, Siredy, Laconbe and others, and which recently have received such support from the statistics collected by Thayer. More gentlemen as a result of careful observations believe that typhoid fever plays an important part in the etiology of arterio-sclerosis; indeed, they believe that it ranks next to acute articular rheumatism. I do not think that the discussion of this topic can be closed more fitly than by quoting the conclusion of Laconbe: "The disorders of the heart appearing some years after recovery from typhoid fever may be legitimately ascribed to this disease, if no other malady capable of comprising the integrity of the heart, either before or after the typhoid, has occurred."

The object of these two lectures has been to emphasize some features of myocarditis that very frequently appear to receive too little consideration; especially is this the case in the first three groups of cases discussed where an increased knowledge of the errors of metabolism will doubtless enable us to retard if we cannot remove the degenerative process. The researchers of the physiological and clinical laboratories promise to put our treatment upon a broader basis, so that in treating cardiac cases the patient will not so often be regarded as if he were only a heart, the patient with Bright's disease as if he were a large kidney, and he will be recognized as a man, an aggregation of organs so interdependent that no one can be affected without influencing another, it may be even to such an extent that the secondary symptoms outclass both in prominence and gravity those of the original lesion.

CHRONIC OTORRHEA AS VIEWED BY THE LIFE INSURANCE COMPANIES AND THE MEDICAL RECRUITING OFFICER.*

By A. BARKAN, M. D., San Francisco.

Motto: "As long as a discharge from the ear exists, we are never able to say how, when or where it may end, nor to what it may lead."—Wilde.

THE RUNNING ear, having been considered harmless, nay a benefit to the general economy of the body, by many from time immemorial, has ceased to be a *noli me tangere*. The last twenty years have brought about a change so radical in the understanding of this malady that radical operative measures have been adopted to fight it. While there is a consensus of opinion as to the indications of thoroughgoing curative methods—nearly all surgical in nature—in many cases of acute and chronic purulent involvment of the temporal bone, the battle still wages with regard to those cases where persons have gone on with ears dry at times, as the patient thinks, slightly discharging at others, with good drainage through a largely perforated drum, with little or no odor to the discharge, no pain or dizziness, and a fair amount of hearing—in a word, then, with a periodically and "comfortably" running ear. Such conditions might be found in the head of a surgeon who does not look with favor upon running sores near any other cavity of the body, in the temporal bone of the monarch, "whose head uneasy lies," with the fear of possible otitic brain complications, or in the ear of a street urchin exposed to all sorts of weather and health conditions.

The profession is divided in rating the seriousness of such conditions. In a discussion on this topic in our local society of eye and ear surgeons a suggestion that some light might be obtained from the rate and death table of life insurance companies led to some personal interviews, and to looking up the topic in literature, principally from this point of view, and as the matter is of some interest also to the general practitioner, I presume to give you the results of my somewhat hurried and fragmentary investigations:

The medical officers of three of the most prominent life insurance companies were interviewed. I elicited from the first and second the following:

"Simple purulent otorrhea, if intermittent and trivial only, is not regarded; but if considerable, or even when slight, if persistent, it imposes an addition to the premium rate equal to one-half per cent of the amount of the insurance. Persistent otorrhea, where the secretion is greenish in color, or offensive in odor, or where there is a history of the occasional discharge of blood, gritty matter or of spiculæ of bone, disqualifies during continuance, and ordinarily for a minimum term of one year after apparent final disappearance. In all cases of otorrhea, therefore, the examiner should make the necessary investigation to cover the foregoing points, and should recite the result in his report."

These are instructions taken from Keating's Handbook. From the third life insurance company I elicited that "chronic otorrhea, when existing, or until two years after it has finally ceased, and proper explanation has been made by the examiner, absolutely excludes." This rule was amended later on as follows: "Cases with otorrhea may be accepted if the disease is unilateral, and a competent aurist furnishes a certificate stating that the perforation is in the lower part of the membrana tympani, and that there is absence of granulations, polypi, caries or involvment of the mastoid cells." Dr. Taylor of the New York Life Insurance Company kindly handed me the February number of the *Medical Examiner and Practitioner*, which had just come into his hands. It contains an article of Dr. Phillips, a New York author-

*Read at the Thirty-fourth Annual Meeting of the State Society, Paso Robles, April 19-21, 1904.